

PETROL-VAPOUR POISONING

BY

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Accidental death due to inhalation of petrol vapour is surprisingly rare in Britain (Aldin, 1958). Nevertheless, it is important to consider the possibility of petrol poisoning whenever illness or death occurs in patients who have been exposed to petrol. The following accident illustrates the danger of spilling petrol inside a small enclosed space.

Case Report

A boy aged 3 years 10 months was trapped in an overturned saloon car. He was discovered 5–10 minutes after the accident lying head downwards, unconscious, with his head in a pool of petrol. He died two minutes after his release without regaining consciousness. The petrol had leaked into the roof of the car from the damaged tank in the boot.

Necropsy (40 hours after death).—The body was pale and cyanosed. There were extensive areas over the trunk and limbs, where the superficial epidermis was loose and could be stripped off with ease. The bases of these excoriated areas were pale and the appearances were very similar to those seen in macerated stillbirths. In addition, there were scattered abrasions and a bruise in the left parietal region of the scalp. The heart and great vessels contained dark fluid blood. There were no petechial haemorrhages in the pericardium, pleura, or thymus. The lungs showed evidence of congestion, oedema, and intrapulmonary haemorrhage, especially in their dorsal portions. The tracheal and bronchial mucosa was hyperaemic and the bronchi were filled with haemorrhagic fluid. There was conspicuous congestion of the remaining organs, but no other significant abnormality, apart from some oedema of the brain and a strong smell of petrol, which was most obvious in the lungs.

Histological Examination.—Hyperaemia was conspicuous in all the organs examined. In addition the lungs showed considerable oedema and some intra-alveolar haemorrhage and necrosis of alveolar walls. There were occasional petechial haemorrhages in sections of brain. Slight fatty change was present in the parenchymal cells of the liver, especially in the periphery of the lobules, and the Kupffer cells throughout the liver were loaded with sudanophil material. In a section of skin from an excoriated area there was loss of much of the superficial part of the epidermis; in places it remained attached, forming intra-epidermal bullae. There was no evidence of any vital reaction in the underlying dermis.

The presence of petrol in the lungs was confirmed by simple distillation, 195 g. of lung tissue yielding 1 ml. of a fluid resembling petrol. Similar treatment of stomach contents (15 ml.), liver (200 g.), and brain (200 g.) failed to demonstrate a measurable quantity of fluid petrol.

Discussion

Death due to inhalation of petrol fumes is a recognized industrial hazard and is usually due to entering a tank or wagon containing petrol (Browning, 1953). Death due to accidental or suicidal ingestion of petrol is more common.

Petrol, or gasoline, is a petroleum distillate consisting essentially of saturated and unsaturated aliphatic hydrocarbons, but, depending on its source, it may contain varying amounts of cyclic and aromatic hydrocarbons. Aromatic hydrocarbons tend to be more toxic than paraffins, perhaps partly because they are absorbed

more rapidly, while, in general, the irritant and toxic effects of a given series of like compounds increase with increase in molecular weight (Machle, 1941). The small amount of tetraethyl lead added to British petrol is unlikely to play any part in acute poisoning.

Machle (1941) states that the fatal oral dose of petrol for man is approximately 7.5 g. per kg. He does not believe that systemic poisoning can be produced by cutaneous absorption alone, though petrol can be absorbed through the skin. Poisoning by inhalation is much more rapid than by ingestion. Susceptible people may show symptoms after exposure to concentrations as low as 300 to 500 parts per million. The exact amount which will cause death in man in a short time is not known, but amounts in excess of 10,000 parts per million are rapidly fatal to most animals (Machle, 1941). A few minutes' exposure to a high concentration causes coma and death without any respiratory struggle or post-mortem signs of anoxia. Lower concentrations may cause flushing of the face, ataxia, mental confusion, slurred speech, and difficulty in swallowing—a picture similar to that of alcoholic intoxication. Delirium, coma, and tonic and clonic convulsions may occur. The blood-pressure is low and the pulse weak and rapid. Respiration is rapid and shallow. Sudden respiratory and cardiac arrest may occur (von Oettingen, 1958).

The pathological changes produced by petrol are dependant on its irritant action and its lipolytic activity. The lungs show the most constant changes: hyperaemia, petechial haemorrhages, and sometimes gross pulmonary haemorrhages and necrosis of alveolar walls. There may be subserous haemorrhages in the liver, kidney, and spleen, and haemorrhages into the serous cavities (in animals). The liver may show cloudy swelling and fatty changes, and the kidneys oedema and damage to the proximal tubules and glomeruli. Oedema and hyperaemia of the brain is usual, and extravasations of blood may occur. If death occurs within a few hours or minutes of exposure few changes may be seen other than oedema, congestion, and scattered haemorrhages in the lungs, and oedema of the central nervous system.

The case described showed some of these features, the changes in the lungs being striking. The skin lesions are similar to those described by Aidin (1958) in a youth dying of petrol-vapour poisoning. Aidin ascribed them to the effect of petrol-soaked clothing in contact with the skin, and he believed that the process went on after death. This would seem to be the most likely explanation. In the present case the boy's clothes were soaked in petrol; they were removed at another hospital, probably an hour or so after the accident. It is obvious that petrol-soaked clothing should be removed as soon as possible to prevent cutaneous absorption and possible damage to the skin.

Petrol vapour is heavier than air and the concentration in the overturned car in the region of the boy's head must have been far in excess of 10,000 parts per million. No fluid petrol was demonstrated in the stomach, but the possibility of aspiration of some fluid petrol into the lungs cannot be excluded. Such aspiration seems unlikely in view of the absence of damage to the oral and pharyngeal mucosa.

Summary

An account is given of the post-mortem findings in a small boy killed in a road accident. These findings and the circumstances of his death indicated that the primary

cause of death was inhalation of petrol vapour. Interesting skin changes, probably due to the contact of petrol-soaked clothing with the skin after death, are described.

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Medical Memoranda

A Case of Fibrinous Bronchitis

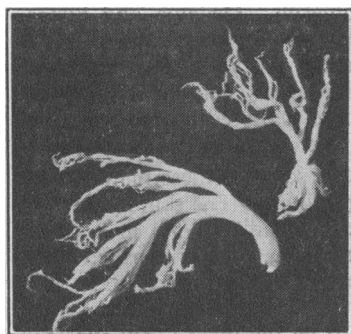
Fibrinous or plastic bronchitis is a rare illness in which attacks of coughing and dyspnoea are associated with the presence of rough fibrinous casts in the bronchial tubes. When the patient coughs up these casts the dyspnoea is relieved. A case of this disorder is described below.

CASE REPORT

The patient, a Turkish youth aged 19, was admitted to the Sisli State Hospital on September 19, 1953, because of pronounced dyspnoea and fever. His illness had started a month previously as a chill, with fever and difficulty in breathing.

On admission he complained of intermittent pain over the praecordium, in the left shoulder, and sometimes in the abdomen, aggravated by inspiration. He was weak, dyspnoeic, had an irritative cough, and assumed a characteristic attitude of distress, sitting up and leaning forward. A friction rub, heard over the praecordium, disappeared shortly after the occurrence of a pericardial effusion. The presence of the effusion was confirmed radiologically.

Over the next few days the pericardial fluid increased. The liver was enlarged, the jugular veins were engorged, and cyanosis of the skin and mucous membranes appeared. Heart sounds were diminished. A pericardial paracentesis was performed and a serofibrinous fluid obtained: it contained much fibrin and a few lymphocytes. A diagnosis of tuberculous pericarditis was made. The fluid was cultured and also injected into a guinea-pig. The culture remained sterile, but the guinea-pig died from tuberculous peritonitis. The patient was treated with corticotrophin. He left hospital on November 25, having recovered. About the middle of January, 1954, he again became dyspnoeic and his face cyanotic. His father noticed that during his struggles for air he obtained relief whenever he coughed up some whitish branched matter. This sputum consisted of tracheo-bronchial casts.



Expectorated casts.

On examination he seemed seriously ill, being dyspnoeic and cyanotic. Chest expansion was moderately limited. Breath sounds were harsh and musical, with numerous moist rales over the base of both lungs. The size of the heart was normal, the sounds were regular, and no abnormality could be detected. No abnormalities were found in other organs. The urine was normal. Blood examination showed

a slight hypochromic anaemia, and there was a slight increase in blood fibrinogen (Dr. Hatice Bodur). Clinically we accepted the case as one of "fibrinous bronchitis following mediastinal compression." X-ray examination (Dr. Tarik Temel) showed diffuse bronchitic changes over the entire lung area, but especially pronounced on the right side. The heart was slightly enlarged.

The white tree-like expectorated casts (see Figure) were examined microscopically (Dr. Fatih Ali Yücel) and were found to be composed of central layers of fibrin, which were oedematous, and the intervening spaces were filled with eosinophils and lymphocytes.

These findings confirmed the clinical diagnosis of fibrinous bronchitis. Aerosol treatment with hyaluronidase was begun in order to dissolve the fibrin. The last radiographs showed improvement in the bronchitic features. The size of the heart was normal.

COMMENT

No satisfactory explanation for this disorder has yet been found. Usually it follows a chronic course and may continue for years. Sometimes it is acute. The patients are mostly young adults or in early middle age, but children may also be affected. The disorder is commoner in males than females.

It occurs chiefly in association with other diseases, especially pulmonary tuberculosis or bronchial asthma, and occasionally during the course of certain acute infections. Nevertheless it may occur as a primary affection with an acute onset in an apparently healthy person. In the chronic form the disorder has a remittent course, with acute or subacute attacks at intervals.

Usually only the signs of ordinary dry bronchitis can be found on examination, but emphysematous changes in the lungs may also occur. The sputum contains tough, whitish masses, varying in size from 1 to 3 cm. in length and about 1 mm. thick, though occasionally they may be larger. They are arranged in concentric layers of material, not very solid and containing air. They consist chiefly of fibrin and mucin and contain eosinophils and neutrophils and occasionally bacteria. In some cases Charcot-Leyden crystals and Curschmann spirals are also found, suggesting a relationship with asthma.

There is a resemblance between the above case and that described by Mulligan and Spencer (1924). Their patient suffered from bronchial asthma, the symptoms of which immediately disappeared after expectoration of fibrinous casts. The cause of the disorder in that case was an enlarged thymus. After x-ray treatment the thymus diminished in size and expectoration of casts ceased. In our case the most probable cause seems to have been mediastinal compression by the pericardial effusion.

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REFERENCE

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Two new standards for sterilizers—B.S. 3219:1960: "Hospital sterilizers: horizontal cylindrical pressure steam type," and B.S. 3220:1960: "Hospital sterilizers: horizontal rectangular pressure steam type"—are now available from the Sales Branch, British Standards Institution, 2, Park Street, London, W.1, price 6s. each (postage extra to non-subscribers).